

Compliments of
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THREE CASES

OF

PRESSURE UPON THE RECURRENT LARYNGEAL
NERVE FROM DIFFERENT CAUSES,

WITH

FIXATION OF THE LEFT VOCAL BAND IN PHONATORY POSITION.

BY

J. SOLIS COHEN, M.D.



REPRINTED FROM THE

TRANSACTIONS OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA,

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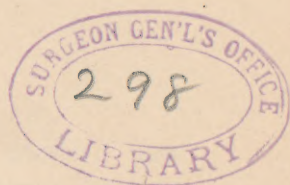
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THE portal for the breath of life is the glottis. Let its two doors become closed, and life becomes extinct within at most two minutes; but let only one of them remain closed never so long, and the interference with respiration may be too slight even to attract attention. The closing of the glottis and its opening are effected by muscles of antagonistic function; yet the opposing innervations are transmitted through fibres of the same nerve trunk, the recurrent laryngeal. Whether this dual innervation proceeds along the same set of fibres from two centres or from one double-acting centre, or whether it proceeds from separate centres along separate sets of fibres traversing the same trunk, as is most

¹ The unusual occurrence of having three such cases under observation at the same time, suggested their presentation for examination and comparison. Death in one instance, and neglect in another, limited the exhibition to one, but the laryngoscopic image was essentially the same in each instance.

plausible, is a problem for which neither experimental nor pathological physiology has yet furnished the solution.

When, therefore, a vocal band, as in the cases herewith presented, stands at continuous rest in the position normally occupied during phonation only, the question arises whether that fixed position is due to tonic spasm or contracture of the muscles which close the glottis on that side, or whether it is the result of paralysis of the muscle which holds the glottis open. Until recently the phenomenon has been attributed solely to neurotic paralysis of the posterior crico-arytenoid muscle, whose function it is with the help of its mate to keep the vocal bands asunder posteriorly, to hold the rima glottidis open for the needs of ordinary respiration, and to draw these bands still further asunder so as to open the glottis to its widest extent for the needs of extraordinary inspirations. This view is supported by the evidence of degeneration of the posterior crico-arytenoid muscle found in almost if not every such case examined *post-mortem*, little or no similar atrophy being discovered in the tissues of the antagonistic muscles. The uniformity with which this atrophic metamorphosis is known to take place, and its attribution to neurotic paralysis of the muscle, have, with other cogent considerations, led an able, accurate, and most painstaking investigator, Dr. Felix Semon, of London, to the conclusion that organic disease of the roots or trunks of the motor nerves of the larynx is likely to become earliest and sometimes exclusively manifested in paralysis of the posterior crico-arytenoid muscle. This view is the one generally entertained and endorsed by laryngologists. An opinion diametrically opposite, on the

other hand, and one with which I am more inclined to accord, has been expressed by Dr. Hermann Krause, of Berlin, based on clinical observation, on neural analogy, and on physiological experiment. In its briefest expression this view maintains that, in analogy with neurotic manifestations in other portions of the body, organic irritation of the recurrent laryngeal nerve produces spasm of all the muscles supplied by its fibres, both those which preside over the patency of the chink of the glottis for respiratory purposes, and those which preside over the approximation of its edges for purposes of phonation, cough, and expulsion of foreign material; but that, inasmuch as the number and mass of muscle and nerve fibres preponderate in the domain for closure, the equilibrium maintained by the respiratory centre under normal conditions is overpowered, so that the spasm remains manifest in the closing phase only, despite the coexistent spasm of the weaker dilator muscle. The opinion held, then, is that the phenomenon of permanent fixation is due to the overpowering contracture or tonic spasm of constricting muscles of the larynx, and not to a paralysis of a dilating one. The atrophy of the dilating muscle is attributed to its mechanical immobility and not to its paralytic immobility. On the other hand, those who believe in primary paralysis of the dilator muscle consider this spasm to be a secondary contracture, and not a primary one. The theory of spasm rather than paralysis, seems to be supported by the first case I had intended to show this evening, but since this paper has been announced, I have learned that the patient died suddenly, away from home, under circumstances that deprived me of the

satisfaction of a projected examination *post-mortem*. Briefly the case is as follows.

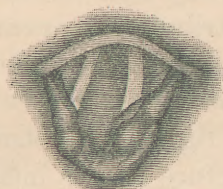
Aneurism of the arch of the aorta and of the left subclavian artery with fixation—spastic immobility?—of the left vocal band in the phonatory position. Mr. X. Y. Z., æt forty-four, formerly a corporal of artillery, and since June, 1865, a machinist, applied November 15, 1885, at the Philadelphia Polyclinic on account of great dyspnœa said to have been but of six months duration, although shortness of breath had existed for fully five years, eventually accompanied with frequent cough, and expectoration of frothy white mucus. Coughing produced great pain within the left side of the thorax. Pain in the left side and in the back had commenced about ten years before, and was always reproduced by excitement, although there was no consciousness of cardiac palpitation. Pains in the epigastric and cardiac areas had existed almost continuously for five years. These were followed by pains radiating along the left arm and up the left side of the neck into the head, where they had remained continuous for the last six months. Dizziness in walking had been produced for some months, compelling interruptions for rest. Dizziness was nearly as bad on standing. About five years before, the voice began to undergo change. It was now weak and diphthonic, the higher pitched tone being metallically shrill and squeaky. Respiration was embarrassed spasmodically on exertion, but during rest in the sitting position was fairly quiet at a rate of from twenty to twenty-four per minute. The patient felt much better in the open air than in a room. The pulse was about 86 per minute at the right wrist and over a pulsatile, expansile tumor below the clavicle and second rib of the left side, but it was almost imperceptible and apparently irregularly intermittent at the left wrist. The pupils were equally dilated.

There was a history of inflammatory rheumatism in 1866, with subsequent histories of intermittent fever and of the primary lesion of syphilis. Appetite and digestion were reported good, with irregularly constipated habit. Emaciation was steadily progressive.

Physical exploration of the thorax revealed aneurism of the arch of the aorta and of the left subclavian artery. Laryngoscopic in-

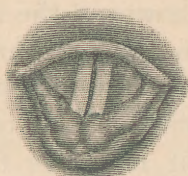
spection revealed tense immobility of the left vocal band in the phonatory position—or rather, with its posterior extremity, a fraction of a line beyond the middle of the glottis, with slightly concave or semielliptic outline of the immovable vocal band (Fig. 1) representing the left half of the ellipse normally produced by the simultaneous action of the two sides in phonation (Fig. 2). The

FIG. 1.



In respiration.

FIG. 2.



In phonation.

aneurism of the aorta compressing the inferior laryngeal nerve as it winds around the arch of that vessel in its recurrent course to the larynx, explained the cause of the fixed position of the vocal band. This fixed position I attributed to preponderating spasm of the group of adductor muscles rather than to paralysis of the single abductor, because of the shrillness of the voice, and the presence of an over-tense vocal band to account for that shrillness or increase of pitch; complete tension requiring contraction and not relaxation of the posterior crico-arytenoid muscle. The patient, who had passed out of my hands, died suddenly while walking in a garden on April 12th, but the manner of death is unknown, whether by spasm of the glottis or by rupture of the aneurism, probably by the latter. The physician summoned after death suspected disease of the heart.

A second patient with a similar permanent fixture of his left vocal band in the phonatory position, is now in the room adjoining. His phonic tone is not shrill as in the other case, but is apparently normal; as is usual in these instances. Consequently, the question as to contracture or paralysis can find no answer in his voice.

There is no aneurism detectable in the chest of this man; but that there may be aneurism pressing posteriorly on the recurrent nerve without physical evidences on auscultation, has been proved not only by a post-mortem examination in a case of my own¹ in which such a correct diagnosis had been made by exclusion in a man with a similar laryngeal defect; but by similar post-mortem discoveries by others. In this case, however, the fixture is not attributed to aneurism, but to another source of irritation, while the inference of contracture is based upon the occasional presence of tremors and even of spasm in the right vocal band, so that at moments the complete picture of the bilateral malady is manifest for a few seconds.

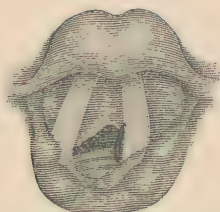
Some eight or more years ago I reported a case of permanent bilateral involvement of this kind preceded by the unilateral lesion. This patient died a few weeks ago, and the structures implicated, muscular and nervous, are undergoing study by Prof. Osler. It is probable that the case before us may become a similar instance of bilateral lesion following a unilateral one. The essential history of the case herewith presented, narrated as briefly as is consistent with its salient features, is as follows:

Fixation of left vocal band in the phonatory position, with tracheal constriction from enlarged thyroid body, and occasional spasm of the mobile vocal band of the right side. X. Y. Z., æt. forty-four, an old private of three years military service; of late years a longshoreman, accustomed to heavy work, lifting large bales of merchandise, applied to the Polyclinic with great dyspnoea and marked tracheal stridor, chiefly inspiratory; with a history of continuous difficulty of breath-

¹ Amer. Journ. Med. Sci., July, 1883, p. 89.

ing preventing sleep, and frequent suffocative attacks of spasm of the glottis especially at night. About one year before, while working actively in a place much exposed to draught, he began to get very hoarse so that he could barely whisper, but he had no fever and no embarrassment in respiration. Cough and expectoration ensued. In about a month his voice began to improve and became gradually restored, but cough and expectoration continued. About August last breathing became embarrassed, became associated with wheezing, and attended with a sense of weight at the epigastrium. This embarrassment in respiration steadily augmented. Just before applying at the Polyclinic, dysphagia became added to his symptoms, and he almost choked when taking food whether solids or liquids. The great stridor made laryngoscopic inspection the first step in investigation. The left vocal band was found fixed in the phonatory position. (Fig. 3.) Both bands were congested and thickened. The right

FIG. 3.



Fixation of left vocal band in phonatory position, with involution of tracheal wall of same side.

band and other laryngeal structures moved freely and normally in respiration and phonation, and on phonation even the supra-arytenoid cartilage of the affected side became bent inward and forward. The great stridor could not be accounted for by the encroachments upon the open glottis, for one-half was entirely normal in proportions and presented ample room for free respiration. Pressure on the trachea was, therefore, suspected, and further inspection revealed an involution of the tracheal wall on the left side and extending anteriorly. External examination of the neck revealed an indurated enlargement of the left side of the thyroid gland of the size of a walnut. The inference following was, that the left lobe of the thyroid body dipped

down so as to press both upon the trachea and upon the recurrent nerve; hence the immobile condition of the left vocal band, and the tracheal stridor. Tendon reflexes were normal. There was slight bilateral relaxation of the palate which did not respond to titillation. Physical examination of the chest revealed small scattered bilateral areas of bronchial breathing most marked along the course of the primitive bronchi. These were attributed to the interruption of the normal tracheal sounds by the involution of the tube acting as a tumor or foreign body. Emphysema existed at the upper part of the left lung, most marked behind. The cardiac impulse was felt at the epigastrium. There was some insufficiency of the mitral valve. No evidence of aneurism could be elicited.

The family and personal history were negative. Nevertheless twenty grain doses of potassium iodide were given three times a day, with marked benefit from the very first; and within a few days dysphagia ceased, tracheal stridor ceased, dyspnoea disappeared except on exertion, and the thyroid gland underwent resolution until now its enlargement is hardly patent to inspection though distinctly perceptible to palpation, especially during movements of deglutition. Involution of the tracheal wall can still be made out on laryngoscopy; the left vocal band is still fixed in the middle line, but the mobility of the supra-arytenoid cartilage has apparently ceased. Within a fortnight or so I have noticed on several occasions that, under the excitement of instructions to phonate strongly or rapidly, or to make deep and rapid inspirations, the hitherto unimpaired vocal band of the right side is sometimes tremulously inobedient to the will, and that once in a while it occupies the middle line for a few seconds despite the strongest efforts at inspiration, which, in fact, increase the difficulty, and produce that characteristic stridor in inspiration which accompanies the bilateral condition under consideration. Certainly the temporary closure of the glottis is at present due to clonic spasm of the adductors of the hitherto sound side of the larynx. Whether this participation of the sound side is indicative of the encroachment of a central lesion which has hitherto been unilateral, or whether it is indicative of reflex motor disturbance from involvement of the sensory fibres of the pneumogastric of the side primarily affected, a local lesion first affecting the recurrent fibres only and

subsequently the other fibres of the pneumogastric, is a question for future study. The only abnormal physical thoracic signs at present are bronchial respiration in the upper part of the chest, with audible perception of the entire phase of expiration over the right bronchus posteriorly, prolonged expiration, as it is usually termed. This I am inclined to attribute simply to resonant deflection of the current against the involuted wall of the trachea. It is possible, however, that an aneurism may be pressing on the trachea, on the bronchi, on the recurrent, and even on the pneumogastric. This single lesion would account for all the phenomena, permanent and evanescent, except the dysphagia and the relaxation of the palate, and even for the relief under iodide of potassium. But I cannot detect such an aneurism. On the other hand, irritative lesion of the cortical centre, or of its communicating fibres with the medulla, or of the vago-accessorius nuclei would account for spasmodic contractions on one side, and the occasional tremors and momentary spasms of the other. The inward movement of the supra-arytenoid cartilage may be explained on the theory of additional innervation from the superior laryngeal, which, according to some authors, is said to innervate the arytenoideus muscle proper.

A third instance of fixation of the left vocal band, due to another cause, recently came under my notice accidentally, as indeed have several instances before.

Fixation of left vocal band in phonatory position, following left-sided pleuritic effusion with displacement of the heart. A young gentleman, nineteen years of age, was sent to me recently for inquiry as to the cause of a hoarseness of several weeks' duration. In addition to evidences of a passing laryngitis, which had produced the hoarseness, I found the left vocal band fixed in the middle line as in the other cases narrated. The hoarseness has passed away, voice is nearly normal, but the vocal band remains immobile in the median line. There is a history of left-sided pleurisy three years ago with effusion enough to push the heart to the right side. The region of the heart is rather prominent, there is slight dulness just below the

upper third of the sternum, the sound of the tricuspid valve is loudest just to the right of the sternum, and the other physical signs so far as I can detect them are of negative value. The theory at present entertained, for the case has not been thoroughly studied out, is that the displacement of the heart to the right and its probable hypertrophy have caused a strain upon the recurrent nerve by dragging the arch of the aorta forward, downward, and to the right, which has either developed a neuritis resulting in neuropathic contracture, or some adhesions of connective tissue or enlarged bronchial glands. Pressure by such swollen glands might induce such a neuritis. In either instance the physical condition of the larynx has nothing to do with the cause of the hoarseness on account of which the interior of the organ was originally inspected.

This case illustrates that when one vocal band is fixed in the middle line, the condition, however serious may be the lesion which has occasioned it, may remain undetected indefinitely. It does not interfere with respiration, for half a glottis is amply sufficient for that function. It does not impair the voice, for the immobile band is in the very best position for good phonation.

Given a vocal band fixed in the middle line in consequence of irritation upon the recurrent nerve, would this be due to spasmodic contracture of the entire group of muscles, or to simple over-action of the adductor portion of that group, because the abductor has become paralyzed?

That tremor precedes spasm is seen in the second case narrated; analogically to what has been witnessed in experiments upon the recurrent nerves, as performed by Krause and others, when fibrillary tremors precede the well-known traction of the vocal band to the middle line.

The great office of the laryngeal nerves is to secure space for respiration, their auxiliary office is to provide means for phonation and cough. While it seems, *a priori*, suitable that the importance of the dilators of the glottis should have caused them to be invested with special immunity, rather than with special vulnerability, it may be argued, on the other hand, that as the main office of the motor laryngeal nerves is to maintain the patency of the glottis for breathing purposes, serious injury to those nerves would compromise the integrity of that office. The reply to this argument would be, that an injury completely destructive of the conductivity of the nerve, leaves the vocal band immobile in a position which does not interfere with the respiratory function.

A hypothetical explanation for these apparent contradictions based upon general physiological principles is offered by my brother Dr. Solomon Solis Cohen. It adopts, in a modified form, the as yet undemonstrated theory of the existence of a cortical motor centre for the larynx, and assumes that the respiratory (abductor) fibres of the recurrent nerve offer less resistance to the passage of nerve-currents than do the phonatory (adductor) fibres; an assumption made highly probable by the ascertained laws of nerve-resistance. The direct energization of a muscle of a limb, it is well known, proceeds *via* an anterior nerve-root from the motor cells of the gray matter of the spinal cord—which may be compared to the local battery in a telegraph line—while the impulse which liberates this energy in obedience to the will, proceeds from a centre for voluntary motion in the cerebral

cortex—the line battery. It may also be set free by so-called reflex action, under the stimulus conveyed from the periphery by an afferent nerve; the peripheral impression becoming the line battery. Applying these facts and hypotheses to the larynx, the following conclusions are drawn. The local battery for laryngeal motion is the nucleus of the spinal accessory nerve in the fourth ventricle. From this proceed two sets of fibres, one, of low resistance, to the dilator muscles, another, of higher resistance, to the constrictor muscles. The ordinary dilatation of the glottis for respiration, which is accomplished by a partial contraction of the dilator muscles, is an automatic function of organic life, under the control of the respiratory centre in the medulla, and takes place with the liberation of the minimum amount of energy by the central motor cells. The respiratory impulses continuously traverse the recurrent nerve, and all other impulses become superimposed upon them. The effect of a first additional increment of nerve-force, which may be set free under psychic impulse from the cortex, is to stimulate further the dilator muscles, opening the glottis wider or to its full extent, as in forced inspiration. More intense psychic stimulation, awakening sufficient energy to overcome the resistance of the adductor fibres of the recurrent nerve, elicits in addition to the continuing action of the dilator muscles (which is necessary to complete the tension of the vocal bands posteriorly) the response of the constricting muscles, and the vocal bands are brought into position for phonation. Reflex stimulation of the centre by an afferent nerve, excites the coughing action of the constricting muscles.

If this explanation of the normal action of the vocal bands be correct, the positions they assume under pathological conditions become easily explicable.

An irritative lesion of the nerve tract, or of the centre, or such lesion of the cortex or of the (hypothetical) cortico-medullar tract as should remove psychic control from nuclear cells continuing to generate nervous force, would produce according to its degree, at one extreme, increased action of the dilating muscle (position of forced inspiration, extreme abduction), or at the other extreme, spasm of all the muscles, bringing the vocal bands into the phonatory position (complete adduction) and between these extremes would be the various manifestations of tremor, spasmodic cough, and even equilibrium (non-paralytic) in the cadaveric position.

In depressive lesions, when the nervous current is too weak to overcome the resistance of the adductor nerve fibres—as when the will fails in hysteria—or when there is partial interference with the generation or transmission of nerve-currents, as in some cases of disease or injury of the nerve tract, the constricting muscles would receive little or no influence, the dilating muscles would continue to act, and there would be presented the well-known picture of adductor paralysis, best exhibited in hysterical aphonia. In hysteria, perfect cough is still possible under reflex stimulation. In organic diseases presenting the same picture, cough should be impaired, but not absent, as organic disease of this nature is not likely to be bilateral.

Destructive lesions of tract or nucleus, if complete, cut off entirely the nervous stimulation; all the muscles

fail to act; and the vocal bands are left in the cadaveric position (paralytic, complete relaxation).

This hypothesis, for the sake of simplicity, omits to take account of normal or pathological inhibition of respiratory acts, or of a possible complication offered by the unknown relations of the sympathetic system with the laryngeal function of respiration.

